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Chapter 20: Thyroid Disorders

## INTRODUCTION

- *Thyroid disorders* involve thyroid hormone production or secretion, altering metabolic stability.

## THYROID HORMONE PHYSIOLOGY

- The synthesis of thyroid hormones, thyroxine ($T_4$) and triiodothyronine ($T_3$), involves four key molecules: iodide, thyroglobulin (TG), thyroid peroxidase, and hydrogen peroxide ($H_2O_2$).
- $H_2O_2$ oxidizes thyroid peroxidase into its active form, which oxidizes iodide ions transported into thyroid follicular cells. The oxidized iodide cells bind to TG, creating iodinated TG.
- Iodinated tyrosine residues—monoiodotyrosine (MIT) and diiodotyrosine (DIT)—couple to form iodothyronines: two molecules of DIT form $T_4$, and MIT and DIT form $T_3$.
- $T_3$, $T_4$, and iodide are released from TG via proteolysis. Iodide is recycled, creating an intrathyroidal iodide reservoir. $T_3$ and $T_4$ are released into the bloodstream by transporters.
- $T_4$ is secreted solely from the thyroid, but <20% of $T_3$ is produced there; most $T_3$ forms from $T_4$ breakdown catalyzed by 5′-monodeiodinase in peripheral tissues. $T_3$ is four times more active than $T_4$. $T_4$ may also be converted by 5′-monodeiodinase into reverse $T_3$, which lacks significant biologic activity.
- Thyroid-stimulating hormone (TSH) from the anterior pituitary regulates thyroid hormone production, controlled by negative feedback of circulating free thyroid hormone and the positive influence of hypothalamic thyrotropin-releasing hormone (TRH). Thyroid hormone production is also regulated by extrathyroidal deiodination of $T_4$ to $T_3$, affected by nutrition, nonthyroidal hormones, drugs, and illness.

## THYROTOXICOSIS (HYPERTHYROIDISM): CLINICAL PRESENTATION

- Symptoms of thyrotoxicosis include nervousness, anxiety, palpitations, emotional lability, easy fatigability, heat intolerance, weight loss despite increased appetite, increased bowel movement frequency, proximal muscle weakness (eg, climbing stairs, arising from sitting position), osteoporosis, and scanty or irregular menses in women.
- Physical signs include warm, smooth, moist skin and unusually fine hair; onycholysis (nail separation from nail beds); eyelid retraction and lid lag (lagging of the upper lid behind the globe upon downward gaze); resting tachycardia, widened pulse pressure, and systolic ejection murmur; fine tremor of the protruded tongue and outstretched hands; hyperactive deep tendon reflexes; and usually thyromegaly.
- Graves' disease presents with hyperthyroidism, diffuse thyroid enlargement, and extrathyroidal findings of exophthalmos, pretibial myxedema, and thyroid acropachy. In severe disease, a thrill may be felt, and a systolic bruit may be heard over the gland.
- In subacute thyroiditis, patients have severe pain in the thyroid region, which often extends to the ear. Systemic symptoms include fever, malaise, myalgia, and signs and symptoms of thyrotoxicosis. The thyroid gland is firm and exquisitely tender on physical examination.
- Painless thyroiditis has a triphasic course that mimics painful subacute thyroiditis. Most patients have mild thyrotoxic symptoms, with lid

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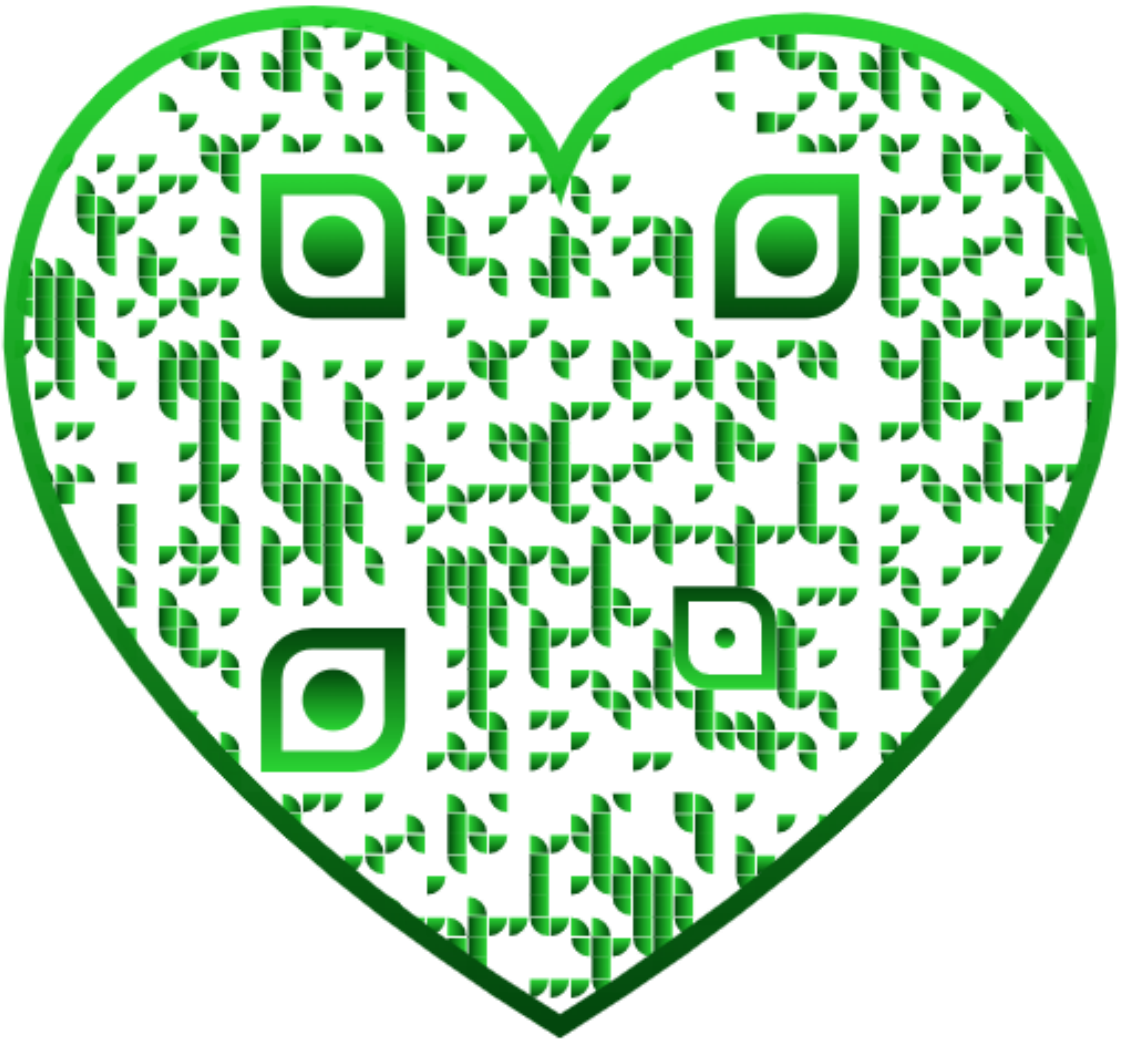

retraction and lid lag but no exophthalmos. The thyroid gland may be diffusely enlarged without tenderness.

- Thyroid storm is a life-threatening emergency characterized by decompensated thyrotoxicosis, high fever (>39.4°C [103°F]), tachycardia, tachypnea, dehydration, delirium, coma, nausea, vomiting, and diarrhea. Triggers include infection, trauma, surgery, radioactive iodine (RAI) treatment, and antithyroid drug withdrawal.

## DIAGNOSIS

- Elevated 24-hour RAI uptake (RAIU) indicates true hyperthyroidism—overproduction of $T_4$, $T_3$, or both (normal RAIU 10%–30%). A low RAIU, without iodine excess, indicates that excess thyroid hormone is likely due to thyroiditis or exogenous thyroid hormone ingestion rather than thyroid gland hyperfunction.
- In thyrotoxic Graves' disease, overall hormone production rate increases with a disproportionate increase in $T_3$ relative to $T_4$ (**Table 20-1**). Thyroid-binding globulin (TBG) becomes saturated due to elevated $T_4$ and $T_3$, reflected by elevated $T_3$ resin uptake. Free $T_4$ and free $T_3$ are increased more than total $T_4$ and total $T_3$. TSH is undetectable due to the negative feedback of thyroid hormone elevation at the pituitary. In symptomatic patients, measuring free $T_4$, total $T_4$, total $T_3$, and TSH confirms thyrotoxicosis. Thyrotropin receptor antibody (TRAb) testing may be useful in nonnodular thyroid and mild/no exophthalmos. If not pregnant or lactating, 24-hour RAI should be obtained when diagnostic uncertainty exists (eg, recent onset of symptoms).
- For toxic adenomas, isolated $T_3$ elevation may occur with autonomously functioning nodules; a $T_3$ must be measured to rule out $T_3$ toxicosis when $T_4$ is normal. If autonomous function is suspected but TSH is normal, diagnosis is confirmed by failure of the nodule to decrease iodine uptake during exogenous $T_3$ administration sufficient to suppress TSH.
- In multinodular goiters, thyroid scan shows patchy areas of autonomously functioning thyroid tissue.
- TSH-induced hyperthyroidism is diagnosed by peripheral hypermetabolism, diffuse thyroid gland enlargement, elevated free thyroid hormone, and elevated TSH. Because the pituitary gland is extremely sensitive to free $T_4$, a "normal" or elevated TSH indicates inappropriate TSH production.
- TSH-secreting pituitary adenomas are diagnosed by an absent TSH response to TRH stimulation, inappropriate TSH, elevated TSH α-subunit, and radiologic imaging.
- In subacute thyroiditis, thyroid function tests follow a triphasic course. Initially, $T_4$ is elevated from the release of preformed thyroid hormone; 24-hour RAIU is <2% because of thyroid inflammation and TSH suppression by elevated $T_4$. As intrathyroidal hormone stores deplete, patients may become mildly hypothyroid with elevated TSH. Recovery follows as thyroid hormone stores replenish, and TSH normalizes.
- In painless thyroiditis, the thyrotoxic phase shows suppressed 24-hour RAIU (<2%), and antithyroglobulin and antithyroid peroxidase antibody are elevated in 50% of patients.
- Thyrotoxicosis factitia is suspected when thyrotoxicosis occurs without increased hormone production, thyroidal inflammation, or ectopic thyroid tissue. RAIU is low from exogenous thyroid hormone suppressing thyroid gland function, and plasma TG is very low.

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TABLE 20-1

**Thyroid Function Tests in Different Thyroid Conditions**

| | Total $T_4$ | Free $T_4$ | Total $T_3$ | TSH |
|---|---|---|---|---|
| Normal | 4.5–10.9 mcg/dL | 0.8–2.7 ng/dL | 60–181 ng/dL | 0.5–4.7 milli-international units/L |
| Hyperthyroid | ↑↑ | ↑↑ | ↑↑↑ | ↓↓[a] |
| Hypothyroid | ↓↓ | ↓↓ | ↓ | ↑↑[a] |
| Increased TBG | ↑ | Normal | ↑ | Normal |

[a]Primary thyroid disease.

## TREATMENT

- Goals of Treatment: Eliminate excess thyroid hormone; minimize symptoms and long-term consequences; and individualize by type and severity of disease, patient age and sex, comorbidities, and prior treatment response.

### NONPHARMACOLOGIC THERAPY

- Thyroidectomy should be considered for large glands (>80 g), severe ophthalmopathy, suspected or proven thyroid malignancy, or coexisting primary hyperparathyroidism requiring surgery. It is also an option when antithyroid drugs fail to induce remission.
- If thyroidectomy is planned, methimazole is given until biochemical euthyroidism (usually 6–8 weeks), followed by iodides for 10–14 days before surgery to decrease gland vascularity.
- **Propranolol may be used for several weeks preoperatively and 7–10 days postoperatively to maintain pulse <90 beats/min. Combined pretreatment with propranolol and 10–14 days of potassium iodide is recommended.**
- Daily plasmapheresis until normal thyroid function is achieved can prepare patients for RAI if antithyroid drugs cause side effects or are ineffective; it can also be used as salvage therapy in thyroid storm.

### PHARMACOLOGIC THERAPY

#### Thionamides

- **Methimazole and propylthiouracil inhibit thyroid hormone synthesis by blocking the thyroid peroxidase enzyme system, preventing trapped iodide oxidation and subsequent organification, and inhibiting coupling of MIT and DIT to form $T_4$ and $T_3$. Propylthiouracil also inhibits peripheral conversion of $T_4$ to $T_3$.**
- Initial doses: methimazole 30–60 mg/day in two to three divided doses or propylthiouracil 300–600 mg/day in three to four divided doses; both may be given once daily.
- Symptom and lab improvements usually occur within 4–8 weeks, at which time tapering to maintenance can begin. Adjust monthly, as $T_4$ steady-state concentrations require this interval. Maintenance: methimazole 5–30 mg/day and propylthiouracil 50–300 mg/day. Continue 12–24 months for long-term remission.

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- Monitor patients every 6–12 months after remission. If a relapse occurs, RAI or surgery is preferred over a second antithyroid drug course, but long-term low-dose methimazole may be considered.
- Minor adverse reactions: pruritic maculopapular rashes, arthralgias, fever, and transient leukopenia (white blood cell count <4000/$mm^3$ or 4 × $10^9$/L). Switching to the alternate thionamide is possible, but cross-sensitivity occurs in about 50% of patients.
- Major adverse reactions: agranulocytosis (with fever, malaise, gingivitis, oropharyngeal infection, and granulocyte count <250 to 500/$mm^3$ or 0.25 to 0.5 × $10^9$/L), aplastic anemia, lupus-like syndrome, gastrointestinal intolerance, pancreatitis, hepatotoxicity, and hypoprothrombinemia. Agranulocytosis usually develops within 3 months; routine white blood cell count monitoring is not recommended due to sudden onset.
- Because of hepatotoxicity risk, propylthiouracil is not first-line in either adults or children, except in (1) the first-trimester pregnancy (when methimazole-induced embryopathy risk exceeds propylthiouracil-induced hepatotoxicity risk), (2) methimazole intolerance, and (3) thyroid storm.

### Iodides

- **Iodide acutely blocks thyroid hormone release, inhibits thyroid hormone biosynthesis by interfering with intrathyroidal iodide use, and decreases gland size and vascularity.**
- Symptoms improve within 2–7 days; $T_4$ and $T_3$ may be reduced for a few weeks.
- Iodides are often used as adjunctive therapy before Graves' disease surgery, to inhibit thyroid hormone release and quickly attain euthyroid state in thyrotoxic patients with cardiac decompensation, or to inhibit thyroid hormone release after RAI.
- **Potassium iodide is available as a saturated solution (SSKI, 35–50 mg iodide/drop) or Lugol's solution (6.25 mg iodide/drop).**
- Starting dose of SSKI is 3–10 drops daily (150–500 mg) in divided doses diluted in water or juice. The dose of Lugol's solution is 5–7 drops (31.25–43.75 mg) three times daily in water or juice. For surgery, administer 7–14 days preoperatively.
- As an RAI adjunct, give SSKI 3–7 days after—not before—RAI to allow thyroid uptake.
- Adverse effects: hypersensitivity (skin rashes, drug fever, and rhinitis, conjunctivitis), salivary gland swelling, stomach upset, diarrhea, nausea/vomiting. "Iodism" (metallic taste, burning mouth and throat, sore teeth and gums, head cold symptoms, and sometimes stomach upset and diarrhea) can occur with prolonged treatment and higher doses. Contraindicated in toxic multinodular goiter, as autonomous tissue utilizes iodine for thyroid hormone synthesis.

### Adrenergic Blockers

- β-Blockers ameliorate palpitations, anxiety, tremor, and heat intolerance, but do not achieve euthyroidism, reduce TRAb, or prevent thyroid storm. Propranolol and nadolol partially block $T_4$ to $T_3$ conversion, but this effect is small.
- Used adjunctively with antithyroid drugs, RAI, or iodides for Graves' disease, toxic nodules, preparation for surgery, or thyroid storm. They are primary therapy only in thyroiditis-related thyrotoxicosis.
- **Propranolol 20–40 mg orally four times daily usually controls adrenergic symptoms (goal heart rate <90 beats/min). Younger or more severely toxic patients may require 240–480 mg/day due to increased clearance.**
- Contraindications: decompensated heart failure (unless caused solely by tachycardia), sinus bradycardia, concomitant monoamine oxidase inhibitors or tricyclic antidepressants, or spontaneous hypoglycemia. Adverse effects include nausea, vomiting, fatigue, insomnia, lightheadedness, and bradycardia. Taper slowly to avoid withdrawal and thyroid storm.
- Centrally acting sympatholytics (eg, clonidine) and calcium channel antagonists (eg, diltiazem) are alternatives if β-blockade is contraindicated.

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### Radioactive Iodine

- **Sodium iodide–131 is a colorless, tasteless, oral liquid that concentrates in the thyroid, initially disrupting hormone synthesis by incorporating into thyroid hormones and TG. Over weeks, affected follicles surrounding tissue undergo necrosis and interstitial fibrosis.**
- RAI is first-line for Graves' disease, toxic autonomous nodules, and toxic multinodular goiters. It is contraindicated in pregnancy due to fetal radiation exposure.
- β-Blockers are the primary adjunctive therapy, usable anytime without interfering with RAI.
- Iodides should be administered 7 days after RAI to avoid interfering with thyroid uptake.
- Patients with cardiac disease and the elderly often receive methimazole before RAI because thyroid hormone transiently increase after RAI treatment from release of preformed thyroid hormone. In cardiac disease, antithyroid drugs may need restarting. They are usually stopped 4–6 days before RAI and restarted 4 days after treatment.
- Administering antithyroid drug therapy immediately after RAI may result in a higher rate of posttreatment recurrence or persistent hyperthyroidism.
- The goal of therapy is destruction of overactive thyroid cells; a single 4000–8000 rad (40–80 Gy) dose achieves euthyroidism in 60% of patients in ≤6 months. A second dose may be given if hyperthyroidism persists after 6 months.
- Hypothyroidism often develops months to years after RAI. The short-term effects include mild thyroidal tenderness and dysphagia. Long-term data show no increased risk of mutations or congenital defects.
- Educate patient on radiation precautions. The American Thyroid Association provides safety recommendations, with restrictions on travel, home, and work/school lasting days to week based on RAI dose.

## TREATMENT OF THYROID STORM

- Promptly initiate: (1) suppression of thyroid hormone formation and secretion, (2) antiadrenergic therapy, (3) corticosteroids, and (4) treatment of complications or precipitating factors (**Table 20-2**).
- **High-dose propylthiouracil is often preferred because it blocks peripheral conversion of $T_4$ to $T_3$ and interferes with thyroid hormone production; however, β-blockers and corticosteroids serve the same purpose. Methimazole's longer duration of action offers a theoretical advantage.**
- **Iodides, which rapidly block release of preformed thyroid hormone, should be administered at least 1 hour after starting thionamide to inhibit iodide utilization by the overactive gland.**
- Short-acting esmolol is the preferred antiadrenergic therapy in the intensive care unit because it is safe in patients with pulmonary disease or cardiac failure risk and is rapidly reversible.
- **Corticosteroids are recommended, but benefits likely stem from antipyretic and blood pressure (BP) stabilizing effects rather than adrenocortical insufficiency.**
- Supportive care includes acetaminophen as an antipyretic (avoid aspirin or other nonsteroidal anti-inflammatory drugs, which displace bound thyroid hormone). Use fluid and electrolyte replacement, sedatives, digoxin, antiarrhythmics, insulin, and antibiotics as indicated.

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TABLE 20-2

**Drug Dosages Used in the Management of Thyroid Storm**

| Drug | Regimen |
|---|---|
| **Therapy Directed Against the Thyroid Gland** | |
| Propylthiouracil | 900–1500 mg/day orally in four or six divided doses |
| Methimazole | 90–120 mg/day orally in four or six divided doses |
| Sodium iodide | Up to 2 g/day IV in single or divided doses |
| Lugol's solution | 5–10 drops three times a day in water or juice |
| Saturated solution of potassium iodide (SSKI) | 5–8 drops every 6 hours in water or juice |
| **Therapy Directed Against Peripheral Thyroid Hormone Effects** | |
| Propranolol | 60–120 mg orally every 6 hours |
| Esmolol | 0.25–0.5 mg/kg IV push then 0.05–0.1 mg/kg/min continuous IV infusion |
| **Therapy Directed Against Systemic Decompensation** | |
| Dexamethasone | 5–20 mg/day orally or IV in divided doses |
| Prednisone | 25–100 mg/day orally in divided doses |
| Methylprednisolone | 20–80 mg/day IV in divided doses |
| Hydrocortisone | 100–400 mg/day IV in divided doses |

## EVALUATION OF THERAPEUTIC OUTCOMES

- After starting hyperthyroidism therapy (surgery, thionamides, or RAI), evaluate monthly until euthyroid.
- Assess for signs of continuing thyrotoxicosis or new hypothyroidism.
- If $T_4$ replacement is initiated, aim to maintain free $T_4$ and the TSH in the normal range. Once stable, monitor every 6–12 months.

## HYPOTHYROIDISM: CLINICAL PRESENTATION

- Symptoms of hypothyroidism include dry skin, cold intolerance, weight gain, constipation, lethargy, weakness, poor appetite, fatigue, low energy, sluggishness, headache, impaired memory, decreased concentration, confusion, slowed speech, muscle aches, muscle cramps, stiffness, carpal tunnel syndrome, hoarseness, and impaired hearing. Menorrhagia and infertility are common in women. In children, growth impairment or neurodevelopmental delay can occur.
- Common physical signs include bradycardia, diminished reflexes, coarse skin and hair, and periorbital and facial puffiness. Thyroid exam may be

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normal or reveal a goiter. Other findings include hypertension, anemia, increased cholesterol and triglycerides, decreased cardiac output, cerebellar dysfunction, tongue thickening, peripheral edema, paresthesia, polyneuropathy, slow relaxation of deep tendon reflexes, slowed motor activity, increased creatinine kinase, cold/pale dry skin, brittle nails, and brittle or hair loss.

- Secondary hypothyroidism due to inadequate TSH often shows pituitary insufficiency, such as abnormal menses or decreased libido, or evidence of a pituitary adenoma, such as visual field defects, galactorrhea, or acromegaloid features.
- Myxedema coma (rare consequence of decompensated hypothyroidism) manifests as hypothermia, severe hypothyroid symptoms, and altered sensorium (delirium to coma). Mortality rates of 60%–70% necessitate immediate, aggressive therapy.

## DIAGNOSIS

- Free $T_4$ and $T_3$ are low with elevated TSH.
- A rise in TSH is the earliest sign of primary hypothyroidism.
- In central hypothyroidism, $T_4$ is low with low/normal TSH. In subclinical hypothyroidism, $T_4$ is normal with elevated TSH.
- In autoimmune thyroiditis, thyroid peroxidase and TG antibodies are elevated.

## TREATMENT OF HYPOTHYROIDISM (TABLE 20-3)

- Goals of Treatment: Restore thyroid hormone concentrations in tissue, relieve symptoms, prevent neurologic deficits in newborns and children, and reverse biochemical abnormalities.
- **Levothyroxine (l-thyroxine, $T_4$) is the preferred replacement and suppressive therapy due to stability, low cost, active oral activity, lack of antigenicity, and uniform potency. It provides a pool of thyroid hormone readily converted to $T_3$.**
- For longstanding disease and older individuals without cardiac disease, start levothyroxine 50 mcg daily, increasing after 4–6 weeks based on TSH.
- For older patients with cardiac disease, start 25 mcg/day, increasing by 25 mcg every 4–6 week to prevent cardiovascular stress.
- Average adult maintenance dose is ~125 mcg/day, but wide variability necessitates individualized therapy and TSH monitoring.
- Subclinical hypothyroidism can be observed without treatment. Treatment is considered when TSH ≥10 mU/L or for young and middle-aged individuals with symptoms of mild hypothyroidism.
- Levothyroxine is the drug of choice for pregnant women, with the goal of normalizing TSH to pregnancy normal reference range. For patients on therapy before pregnancy, increase the dose by 25%–30% (two extra tablets) once pregnancy is suspected. Discontinue treatment if first diagnosed during pregnancy and return to prepregnancy doses after delivery, with TSH rechecked 6 weeks postpartum.
- Absorption may be impaired by foods/supplements (iron, calcium, soy, fiber, espresso), over-the-counter medications (aluminum antacids, simethicone, orlistat), heavy metals (chromium, colloidal silver), phosphate binders (sevelamer, lanthanum), bile acid sequestrants (cholestyramine, colesevelam, colestipol), and sodium polystyrene sulfonate, sucralfate, and raloxifene). Acid suppression (histamine blockers and proton pump inhibitors) may also reduce levothyroxine absorption.
- Levothyroxine requirements may increase with: (1) drugs that increase $T_4$ metabolism (diabetes drugs, antiseizure drugs, warfarin, simvastatin, rifampin, rifapentine, imatinib, lopinavir/ritonavir, chloroquine); (2) high estrogen states (oral contraceptives, postmenopausal hormone replacement); (3) blocked $T_4$ to $T_3$ conversion (selenium deficiency, β-blockers, bugleweed herbal, amiodarone); or (4) reduced thyroid hormone production (amiodarone, iodine-containing medications, lithium and herbals kelp and laminaria). Several herbals can decrease levothyroxine requirements.

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- **Thyroid USP (desiccated thyroid), usually derived from pig thyroid, may cause allergies or antibody development. Although inexpensive, its high proportion of $T_3$ increases thyrotoxicosis risk.**
- **Liothyronine (synthetic $T_3$) has uniform potency but more cardiac adverse effects, higher cost, difficult monitoring, dosing requirement of three times a day, and prolonged adjustment to euthyroidism.**
- Excessive doses may lead to heart failure, atrial fibrillation, angina pectoris, myocardial infarction (MI), and coronary artery spasm. Hyperthyroidism reduces bone density and increases fracture risk.

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TABLE 20-3

**Thyroid Preparations Used in the Treatment of Hypothyroidism**

| Drug (Brand Name) | Dosage Form | Content | Relative Dose | Comments/Equivalency |
|---|---|---|---|---|
| Thyroid USP (Armour Thyroid, Adthyza, Nature-Throid, NP Thyroid, Westhroid, WP Thyroid) $T_4$:$T_3$ ratio approximately 4.2:1 | Doses include 1/4, 1/2, 1, 1½, 2, 3, 4, and 5 grain tablets<br>Armour Thyroid and NP Thyroid 1 grain = 60 mg; Nature-Throid, Westhroid, and WP Thyroid 1 grain = 65 mg. | Desiccated pork thyroid gland | 1 grain (equivalent to 74–100 mcg of $T_4$) | High $T_3$:$T_4$ ratio; inexpensive; generic brands may not be bioequivalent |
| Levothyroxine (Ermeza, Euthyrox, Levoxyl, Synthroid, Thyquidity, Tirosint, Tirosint-SOL, Unithroid) | Tablets (eg, Euthyrox, Levoxyl, Synthroid, Unithroid): 25, 50, 75, 88, 100, 112, 125, 137, 150, 175, 200, 300 mcg tablets<br>Liquid-filled oral capsules (eg, Tirosint): 13–2000 mcg<br>Liquid solution (eg, Ermeza, Thyquidity, Tirosint-Sol): 13, 20, 25, 30, 37.5, 44, 50, 62.5, 75, 88, 100, 112, 125, 137, 150, 175, and 200 mcg/mL<br>Intravenous solution: 20, 40, 100 mcg/mL<br>Intravenous powder for solution: 100, 200, 500 mcg | Synthetic $T_4$ | 100 mcg | Stable; predictable potency; available oral tablet, oral capsule, oral solution, intravenous solution, and intravenous powder for solution; generics may be bioequivalent; when switching from natural thyroid to L-thyroxine, lower dose by one-half grain; variable absorption between products; daily dosing (half-life = 7 days); considered to be drug of choice |
| Liothyronine (Cytomel) | Tablets: 5, 25, and 50 mcg tablets<br>Intravenous solution: 0.01 mg/mL | Synthetic $T_3$ | 33 mcg (~equivalent to 100 mcg $T_4$) | Uniform absorption, rapid onset; half-life = 1.5 days, rapid peak and troughs |

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### TREATMENT OF MYXEDEMA COMA

- Immediate/aggressive therapy is IV bolus levothyroxine, 300–500 mcg. IV liothyronine or a combination of both hormones may be used because it impairs $T_4$ to $T_3$ conversion.
- Give IV hydrocortisone 100 mg every 8 hours until adrenal suppression is excluded.
- Consciousness, lowered TSH, and vital sign stabilization are expected within 24 hours.
- Maintenance: levothyroxine 75–100 mcg IV until stable and oral therapy begins.
- Provide supportive therapy to maintain adequate ventilation, euglycemia, BP, and body temperature. Diagnose and treat underlying triggers, such as sepsis and MI.

## EVALUATION OF THERAPEUTIC OUTCOMES

- TSH is the most sensitive and specific marker for levothyroxine dose adjustment. Concentrations fall within hours and usually normalize within 2–6 weeks.
- Monitor TSH and $T_4$ every 4–6 weeks until euthyroid. Elevated TSH indicates insufficient replacement. $T_4$ helps detect noncompliance, malabsorption, or levothyroxine product bioequivalence issues. TSH also aids in identifying noncompliance.
- In hypothyroidism from hypothalamic or pituitary failure, appropriate levothyroxine replacement dose is judged by symptom relief and normalization of $T_4$.

*See Chapter 100, Thyroid Disorders, authored by Kathleen J. Pincus and Rachel W. Khan, for a more detailed discussion of this topic.*

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